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Original Article

Sleep duration and abnormal serum lipids: the China Health and Nutrition Survey

Yiqiang Zhan, Ruoqing Chen, Jinming Yu *

Institute of Clinical Epidemiology, School of Public Health, Fudan University, Shanghai, PR China

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ABSTRACT

Objective: To examine the associations between sleep duration and total cholesterol (TC), triglyceride (TG), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), apolipoprotein B (ApoB), apolipoprotein A1 (ApoA1), and lipoprotein (a) [Lp(a)].
Methods: The present study analyzed 8574 adults from the China Health and Nutrition Survey (2009). Sleep duration was classified into ⩽6, 7, 8, 9, and ⩾10 h. Age, education, occupation, current smoking, current drinking, physical activity, body mass index, hypertension, and diabetes were adjusted as confounders in gender-stratified multiple logistic regression models.
Results: Compared with women reporting 8 h sleep duration, the odds ratios (ORs) and 95% confidence intervals (CIs) of high TC for those with ⩽6, 7, 9, and ⩾10 h were 1.65 (1.32–2.06), 1.19 (1.00–1.43), 1.11 (0.89–1.39), and 1.27 (1.02–1.60) after adjusting for confounders. Likewise, the ORs (95% CIs) of high LDL-C were 1.71 (1.28–2.29), 1.36 (1.05–1.76), 1.04 (0.74–1.46), and 1.09 (0.78–1.53), whereas those of high ApoB were 1.80 (1.34–2.42), 1.15 (0.88–1.52), 0.95 (0.66–1.35), and 1.00 (0.70–1.43) for women with ⩽6, 7, 9, and ⩾10 h sleep duration, respectively. These associations were not statistically significant in men.
Conclusions: Both shorter and longer sleep durations were associated with higher risks of abnormal serum lipid profiles in women but not in men.

1. Introduction

Shorter sleep duration has been increasingly reported to be associated with higher risks of hypertension [1], diabetes [2], psychological disorders [3], and cardiovascular disease mortality [4]. Recently, a study reported that short sleep duration was associated with dyslipidemia in an American adolescent population [5]. However, the result was not consistent with findings from a Chinese population-based study in Taiwan [6], which stated that short sleep duration was unrelated to a high level of triglyceride (TG) or to a low level of high density lipoprotein cholesterol (HDL-C). Additionally, a U-shaped association between sleep duration and metabolic disorders was described in two studies [7,8], both of which argued that shorter or longer sleep duration might be risk factors for abnormal serum lipid levels. However, a study in Norway did not reveal such a relationship between sleep duration and total cholesterol (TC), HDL-C, or TG [9].

Apolipoprotein B (ApoB), apolipoprotein A1 (ApoA1), and lipoprotein (a) [Lp(a)] are also normally measured as serum lipid markers in clinical practice. However, to the best of our knowledge, studies regarding the associations between sleep duration and ApoB, ApoA1, or Lp(a) are scarce. The aim of this study was to investigate the prevalence of abnormal serum lipids [TC, TG, LDL-C, HDL-C, ApoA1, ApoB, and Lp(a)] and their associations with sleep duration using data from the China Health and Nutrition Survey (CHNS) [10], which offered a unique opportunity to explore their relationships and to improve our understanding of sleep and lipids in health.

2. Methods

2.1. Study subjects

The China Health and Nutrition Survey (CHNS) [11] was initiated in 1989, with the aim to understand changes in health status using a follow-up interval of two or three years. The CHNS selected individuals from 228 communities and was designed to represent 56% of China's population from nine provinces including Liaoning, Shandong, Heilongjiang, Henan, Jiangsu, Hubei, Hunan, Guizhou,

* Corresponding author. Address: Institute of Clinical Epidemiology, School of Public Health, Fudan University, 130 Dong'An Road, Shanghai 200032, PR China. Tel./fax: +86 2154237868.
E-mail address: jmy@fudan.edu.cn (J. Yu).

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and Guangxi. A multi-stage, random cluster sampling design was used to draw study samples. This survey was approved by the institutional review committees of the University of North Carolina at Chapel Hill, the National Institute of Nutrition and Food Safety, the Chinese Center for Disease Control and Prevention, and the China–Japan Friendship Hospital, Ministry of Health. All participants provided written informed consent. Details about the study design were reported elsewhere [11].

In the 2009 wave of the CHNS, blood samples were collected and assessed for the first time. In all, 10,242 individuals aged ⩾7 years provided fasting blood and anthropometric measures. In the present study, we excluded those who had missing values of blood sample tests or sleep duration ($n = 853$) and those who were aged <18 years ($n = 815$). Altogether, 8574 adults were included in the analysis.

2.2. Measurements of abnormal serum lipid and sleep duration

Blood samples were collected by venipuncture after an overnight fast. Plasma and serum samples were then frozen, and stored at −86 °C for laboratory analysis. All samples were analyzed in a national central laboratory in Beijing (medical laboratory accreditation certificate ISO 15189:2007) with strict quality control. TC, HDL-C and LDL-C were measured using the glycerol–phosphate oxidase method, and the polyethylene glycol (PEG)-modified enzyme method, respectively, by determiner regents (Kyowa Medex Co., Ltd, Tokyo, Japan). TG was measured using the glycerol–phosphate oxidase method and the PEG-modified enzyme method, by determiner regents (Kyowa Medex Co., Ltd, Tokyo, Japan). ApoB and ApoA1 were assessed using the immunoturbidimetric method, by determiner regents (Randox Laboratories, Ltd, UK). Lp(a) was assessed using the immunoturbidimetric method, by determiner regents (Denka Seiken Co., Ltd, Japan). All lipid measures were on the Hitachi 7600 automated analyzer (Hitachi, Inc., Tokyo, Japan).

Abnormal TC was defined as TC >5.18 mmol/L, abnormal TG was defined as TG >1.70 mmol/L, abnormal LDL-C was defined as LDL-C >3.37 mmol/L, and abnormal HDL-C was defined as HDL-C <1.04 mmol/L. Dyslipidemia was defined by any of the levels of TC, TG, LDL-C and HDL-C being abnormal, in accordance with the Chinese Guidelines on Prevention and Treatment of Dyslipidemia in Adults [12]. Sleep duration was self-reported and categorized into five groups: ⩽6, 7, 8, 9, and ⩾10 h.

2.3. Potential confounders

Blood pressure (BP) was defined by the mean of three measurements after a 10 min seated rest, using standard mercury sphygmomanometers with regular adult cuffs. Hypertension was defined by BP ⩾140/90 mmHg or currently taking anti-hypertension medicines. Diabetes was defined as fasting glucose ⩾126 mg/dL or taking diabetes medication. Body mass index (BMI, kg/m^2), current smoking status, current drinking status, and physical activity were also considered as potential confounders.

Table 1
Characteristics of the male study subjects from the China Health and Nutrition Survey 2009.

Variables	⩽6 h ($n = 386$)	7 h ($n = 761$)	8 h ($n = 1902$)	9 h ($n = 479$)	⩾10 h ($n = 452$)	*P*
Age (years)	56.3 ± 13.6	51.7 ± 13.6	49.1 ± 13.9	50.2 ± 16.4	53.1 ± 18.3	<0.001
BMI (kg/m^2)	23.31 ± 3.64	23.63 ± 3.57	23.39 ± 3.51	23.19 ± 3.50	22.86 ± 3.54	0.007
TC (mmol/L)	4.87 ± 1.07	4.90 ± 1.03	4.82 ± 1.01	4.77 ± 1.03	4.70 ± 1.00	0.008
TG (mmol/L)	1.79 ± 1.44	1.84 ± 1.24	1.83 ± 1.24	1.72 ± 1.20	1.79 ± 1.15	0.791
LDL-C (mmol/L)	2.95 ± 1.00	2.99 ± 0.96	2.92 ± 0.99	2.92 ± 0.96	2.80 ± 1.03	0.021
HDL-C (mmol/L)	1.42 ± 0.37	1.38 ± 0.56	1.40 ± 0.48	1.35 ± 0.39	1.38 ± 0.41	0.289
ApoB (g/L)	0.94 ± 0.29	0.93 ± 0.27	0.90 ± 0.27	0.89 ± 0.27	0.87 ± 0.27	<0.001
ApoA1 (g/L)	1.18 ± 0.34	1.13 ± 0.45	1.14 ± 0.37	1.12 ± 0.33	1.12 ± 0.34	0.267
Lp(a) (g/L)	13.98 ± 26.22	14.59 ± 19.99	13.97 ± 26.9	14.12 ± 21.63	15.1 ± 21.56	0.564
Education						<0.001
<10 years	279 (72.2)	508 (66.7)	1328 (69.8)	359 (74.9)	365 (80.7)	
10–12 years	75 (19.5)	177 (23.3)	434 (22.8)	97 (20.3)	71 (15.8)	
⩾12 years	32 (8.3)	76 (10.0)	141 (7.4)	23 (4.8)	16 (3.5)	
Occupation						<0.001
No job	75 (19.5)	148 (19.4)	348 (18.3)	104 (21.7)	129 (28.5)	
Blue collar	189 (48.9)	393 (51.6)	1103 (58.0)	266 (55.6)	234 (51.8)	
Retired	74 (19.3)	112 (14.7)	204 (10.7)	70 (14.7)	65 (14.4)	
White collar	47 (12.3)	109 (14.3)	247 (13.0)	38 (8.0)	24 (5.3)	
Current smoking						0.147
No	135 (35.0)	292 (38.4)	747 (39.3)	202 (42.2)	161 (35.6)	
Yes	251 (65.0)	469 (61.6)	1155 (60.7)	277 (57.8)	291 (64.4)	
Current drinking						0.008
No	143 (37.0)	279 (36.7)	758 (39.9)	210 (43.8)	206 (45.6)	
Yes	243 (63.0)	482 (63.3)	1144 (60.1)	269 (56.2)	246 (54.4)	
Physical activity						0.043
Not regular	345 (89.4)	653 (85.8)	1689 (88.8)	432 (90.2)	411 (90.9)	
Regular	41 (10.6)	108 (14.2)	213 (11.2)	47 (9.8)	41 (9.1)	
Hypertension						<0.001
No	237 (62.0)	545 (72.2)	1380 (73.5)	344 (72.7)	292 (65.3)	
Yes	145 (38.0)	210 (27.8)	498 (26.5)	129 (27.3)	155 (34.7)	
Diabetes						0.902
No	348 (90.2)	691 (91.0)	1733 (91.3)	432 (90.6)	406 (90.0)	
Yes	38 (9.8)	68 (9.0)	166 (8.7)	45 (9.4)	45 (10.0)	

BMI, body mass index; TC, total cholesterol; TG, triglyceride; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; ApoB, apolipoprotein B; ApoA1, apolipoprotein A1; Lp(a), lipoprotein (a).

Table 2
Characteristics of the female study subjects from the China Health and Nutrition Survey 2009.

Variables	⩽6 h (n = 482)	7 h (n = 869)	8 h (n = 2146)	9 h (n = 529)	⩾10 h (n = 505)	P
Age (years)	59.0 ± 13.6	52.5 ± 13.6	48.3 ± 13.9	50.1 ± 16.4	52.6 ± 18.3	<0.001
BMI (kg/m^2)	23.73 ± 3.64	23.58 ± 3.57	23.41 ± 3.51	23.08 ± 3.50	23.23 ± 3.54	0.029
TC (mmol/L)	5.20 ± 1.07	4.95 ± 1.03	4.84 ± 1.01	4.81 ± 1.03	4.90 ± 1.00	<0.001
TG (mmol/L)	1.78 ± 1.44	1.52 ± 1.24	1.54 ± 1.24	1.53 ± 1.2	1.60 ± 1.15	0.006
LDL-C (mmol/L)	3.27 ± 1.00	3.09 ± 0.96	2.97 ± 0.99	2.92 ± 0.96	3.03 ± 1.03	<0.001
HDL-C (mmol/L)	1.47 ± 0.37	1.49 ± 0.56	1.48 ± 0.48	1.49 ± 0.39	1.48 ± 0.41	0.935
ApoB (g/L)	1.02 ± 0.29	0.93 ± 0.27	0.89 ± 0.27	0.88 ± 0.27	0.90 ± 0.27	<0.001
ApoA1 (g/L)	1.21 ± 0.34	1.20 ± 0.45	1.16 ± 0.37	1.19 ± 0.33	1.18 ± 0.34	0.022
Lp(a) (g/L)	17.76 ± 26.22	15.37 ± 19.99	16.53 ± 26.9	16.00 ± 21.63	17.23 ± 21.56	0.472
Education						<0.001
<10 years	320 (82.8)	575 (75.6)	1472 (77.4)	413 (86.3)	399 (88.2)	
10–12 years	60 (15.5)	151 (19.9)	329 (17.3)	51 (10.7)	47 (10.3)	
⩾12 years	7 (1.7)	42 (5.5)	101 (5.3)	14 (3.0)	7 (1.5)	
Occupation						<0.001
No job	164 (42.5)	246 (32.3)	654 (34.4)	202 (42.1)	240 (53.0)	
Blue collar	123 (31.8)	324 (42.6)	860 (45.2)	207 (43.2)	164 (36.3)	
Retired	86 (22.3)	127 (16.7)	200 (10.5)	48 (10.0)	39 (8.6)	
White collar	13 (3.4)	64 (8.4)	188 (9.9)	23 (4.7)	9 (2.1)	
Current smoking						<0.001
No	434 (90.0)	839 (96.5)	2077 (96.8)	511 (96.6)	486 (96.2)	
Yes	48 (10.0)	30 (3.5)	69 (3.2)	18 (3.4)	19 (3.8)	
Current drinking						0.440
No	432 (89.6)	790 (90.9)	1959 (91.3)	492 (93.0)	461 (91.3)	
Yes	50 (10.4)	79 (9.1)	187 (8.7)	37 (7.0)	44 (8.7)	
Physical activity						<0.001
Not regular	426 (88.4)	773 (89.0)	1970 (91.8)	497 (94.0)	481 (95.2)	
Regular	56 (11.6)	96 (11.0)	176 (8.2)	32 (6.0)	24 (4.8)	
Hypertension						<0.001
No	289 (60.3)	608 (70.5)	1673 (78.5)	400 (76.9)	364 (72.7)	
Yes	190 (39.7)	254 (29.5)	457 (21.5)	120 (23.1)	137 (27.3)	
Diabetes						0.069
No	435 (90.2)	813 (93.6)	2009 (93.9)	493 (93.5)	469 (93.1)	
Yes	47 (9.8)	56 (6.4)	130 (6.1)	34 (6.5)	35 (6.9)	

BMI, body mass index; TC, total cholesterol; TG, triglyceride; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; ApoB, apolipoprotein B; ApoA1, apolipoprotein A1; Lp(a), lipoprotein (a).

Table 3
Prevalence of abnormal serum lipid in men from the China Health and Nutrition Survey 2009 [n (%)].

	⩽6 h	7 h	8 h	9 h	⩾10 h	P
High TC	128 (33.2)	269 (35.4)	610 (32.1)	137 (28.7)	128 (28.4)	0.070
High TG	82 (21.2)	187 (24.6)	427 (22.5)	92 (19.3)	103 (22.8)	0.206
High LDL-C	41 (10.6)	72 (9.5)	180 (9.5)	44 (9.2)	32 (7.1)	0.395
Low HDL-C	170 (44.0)	385 (50.7)	922 (48.6)	239 (50.1)	223 (49.4)	0.287
High ApoB	21 (5.4)	41 (5.4)	82 (4.3)	16 (3.4)	15 (3.3)	0.330
Low ApoA1	16 (4.1)	39 (5.1)	72 (3.8)	10 (2.1)	15 (3.3)	0.056
High Lp(a)	46 (11.9)	89 (11.7)	231 (12.2)	54 (11.3)	70 (15.5)	0.150
Dyslipidemia	258 (66.8)	551 (72.6)	1308 (68.9)	325 (68.1)	303 (67.2)	0.094

TC, total cholesterol; TG, triglyceride; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; ApoB, apolipoprotein B; ApoA1, apolipoprotein A1; Lp(a), lipoprotein (a).

Table 4
Prevalence of abnormal serum lipid in women from the China Health and Nutrition Survey 2009 [n (%)].

	⩽6 h	7 h	8 h	9 h	⩾10 h	P
High TC	235 (48.8)	318 (36.6)	719 (33.6)	170 (32.3)	191 (38.0)	<0.001
High TG	110 (22.8)	134 (15.4)	364 (17.0)	81 (15.4)	105 (20.9)	0.003
High LDL-C	96 (19.9)	117 (13.5)	227 (10.6)	52 (9.9)	58 (11.5)	<0.001
Low HDL-C	52 (10.8)	66 (7.6)	214 (10.0)	46 (8.7)	49 (9.7)	0.217
High ApoB	95 (19.7)	95 (10.9)	197 (9.2)	44 (8.3)	51 (10.1)	<0.001
Low ApoA1	18 (3.7)	20 (2.3)	35 (1.6)	18 (3.4)	13 (2.6)	0.023
High Lp(a)	76 (15.8)	108 (12.4)	300 (14.0)	72 (13.7)	85 (16.9)	0.163
Dyslipidemia	292 (60.6)	403 (46.4)	988 (46.2)	229 (43.5)	258 (51.3)	<0.001

TC, total cholesterol; TG, triglyceride; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; ApoB, apolipoprotein B; ApoA1, apolipoprotein A1; Lp(a), lipoprotein (a).

2.4. Statistical analysis

All statistical analyses were conducted using R 2.15 [13]. For descriptive analyses, percentages were presented for categorical variables, and means plus standard deviations were calculated for continuous variables. The basic characteristics of study subjects, the prevalence of dyslipidemia and the individual abnormal serum lipid components by categories of sleep duration are presented. The Cochran–Armitage method was used for the trend test. The association between sleep duration and abnormal serum lipids was then examined, using multiple logistic regression models with robust estimation of standard errors in all participants. We also tested the interaction terms between sleep duration and the other independent variables in the models, and found that the interaction terms between gender and sleep duration were significant ($P < 0.0001$). Thus, we conducted the analysis for all serum lipid markers in men and women separately when additionally taking account of previous publications. Three different models were used to investigate the association between sleep duration and abnormal serum lipids. The first model only included sleep duration and age followed by the second model adjusted for education, occupation, current smoking, current drinking, and physical activity as confounders. The third model was further adjusted for BMI, diabetes and hypertension. We also stratified the analysis by age ('young': <60 years; 'old': ⩾60 years) (see Supplementary Tables 1–4). Odds ratios (ORs) with 95% confidence intervals (CIs) were presented, and $P < 0.05$ was considered statistically significant.

3. Results

3.1. Basic characteristics of study subjects

The age range was 18–92 years for men and 18–98 years for women. There was no statistically significant difference between men and women in the percentages of participants in different sleep duration categories ($P = 0.693$). Basic characteristics of the study subjects were described in Table 1 for men and in Table 2 for women.

Table 5
Association between sleep duration and serum lipid in men from the China Health and Nutrition Survey 2009 [odds ratio (95% confidence interval)].

Serum lipid	Sleep duration (h)	Model 1[a]	Model 2[b]	Model 3[c]
High TC	⩽6	0.99 (0.77–1.23)	0.95 (0.75–1.21)	0.95 (0.74–1.21)
	7	1.11 (0.93–1.33)	1.06 (0.89–1.28)	1.07 (0.89–1.28)
	8	1	1	1
	9	0.85 (0.68–1.06)	0.88 (0.70–1.10)	0.87 (0.70–1.10)
	⩾10	0.79 (0.62–0.99)	0.84 (0.66–1.07)	0.83 (0.65–1.06)
High TG	⩽6	1.01 (0.77–1.32)	1.01 (0.73–1.29)	1.01 (0.73–1.29)
	7	1.15 (0.94–1.40)	1.07 (0.87–1.32)	1.08 (0.87–1.34)
	8	1	1	1
	9	0.80 (0.62–1.03)	0.82 (0.63–1.08)	0.81 (0.62–1.06)
	⩾10	1.04 (0.81–1.33)	1.16 (0.89–1.51)	1.13 (0.86–1.47)
High LDL-C	⩽6	1.00 (0.69–1.76)	1.00 (0.68–1.76)	1.00 (0.68–1.44)
	7	0.96 (0.72–1.55)	0.92 (0.69–1.55)	0.92 (0.69–1.24)
	8	1	1	1
	9	0.94 (0.66–1.70)	0.97 (0.68–1.70)	0.96 (0.68–1.37)
	⩾10	0.65 (0.43–1.85)	0.69 (0.46–1.86)	0.69 (0.45–1.04)
Low HDL-C	⩽6	0.99 (0.73–1.41)	0.99 (0.72–1.44)	0.99 (0.72–1.17)
	7	1.15 (0.97–1.30)	1.12 (0.93–1.31)	1.11 (0.93–1.33)
	8	1	1	1
	9	1.06 (0.86–1.36)	1.11 (0.89–1.39)	1.10 (0.89–1.37)
	⩾10	1.04 (0.84–1.38)	1.15 (0.92–1.40)	1.14 (0.91–1.43)
High ApoB	⩽6	1.15 (0.69–1.91)	1.13 (0.68–1.88)	1.14 (0.68–1.90)
	7	1.18 (0.80–1.73)	1.12 (0.75–1.65)	1.14 (0.77–1.68)
	8	1	1	1
	9	0.75 (0.44–1.30)	0.78 (0.45–1.35)	0.77 (0.44–1.34)
	⩾10	0.69 (0.39–1.24)	0.74 (0.41–1.33)	0.71 (0.39–1.27)
Low ApoA1				
	⩽6	0.95 (0.53–1.68)	0.90 (0.51–1.61)	0.90 (0.50–1.60)
	7	1.33 (0.89–2.00)	1.34 (0.89–2.01)	1.36 (0.90–2.03)
	8	1	1	1
	9	0.49 (0.24–1.02)	0.48 (0.24–1.02)	0.49 (0.24–1.03)
	⩾10	0.73 (0.40–1.34)	0.79 (0.38–1.28)	0.78 (0.37–1.25)
High Lp(a)				
	⩽6	0.96 (0.67–1.35)	1.01 (0.67–1.36)	1.01 (0.67–1.36)
	7	0.97 (0.74–1.26)	1.00 (0.75–1.29)	1.01 (0.76–1.29)
	8	1	1	1
	9	0.94 (0.68–1.29)	0.92 (0.66–1.27)	0.92 (0.67–1.27)
	⩾10	1.39 (1.03–1.87)	1.31 (0.97–1.76)	1.31 (0.97–1.76)
Dyslipidemia				
	⩽6	0.97 (0.76–1.23)	1.00 (0.76–1.26)	1.00 (0.75–1.25)
	7	1.22 (1.01–1.48)	1.18 (0.96–1.44)	1.18 (0.96–1.44)
	8	1	1	1
	9	0.95 (0.76–1.18)	1.01 (0.80–1.27)	1.00 (0.79–1.26)
	⩾10	0.89 (0.71–1.11)	1.01 (0.80–1.29)	1.00 (0.79–1.27)

TC, total cholesterol; TG, triglyceride; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; ApoB, apolipoprotein B; ApoA1, apolipoprotein A1; Lp(a), lipoprotein (a).

[a] Model 1: sleep duration, age.

[b] Model 2: sleep duration, age, education, occupation, current smoking, current drinking, physical activity.

[c] Model 3: sleep duration, age, education, occupation, current smoking, current drinking, physical activity, body mass index, hypertension, and diabetes.

3.2. Prevalence of abnormal serum lipids

The prevalences of dyslipidemia were significantly different between the sleep duration categories for women ($P > 0.001$) but not for men ($P = 0.094$). The prevalences of other serum lipid markers are summarized in Table 3 for men and in Table 4 for women.

3.3. Association of sleep duration and abnormal serum lipids

Compared with men having 8 h sleep duration, those with lower or higher sleep durations did not exhibit higher risks of abnormal lipid profiles after multivariate adjustments (Table 5). However, the ORs (95% CIs) of dyslipidemia for women with sleep duration of ⩽6 and ⩾10 h were 1.34 (1.07–1.68) and 1.23 (0.99–1.53) compared with 8 h sleep duration after multivariate adjustments (Table 6). Likewise, similar results were observed for high TC, high TG, low LDL-C, and high ApoB in women.

4. Discussion

Various studies have investigated sleep disorder and its consequences during the last decades. Further studies have been published to explore abnormal lipid profile and its risk factors. However, only a few of them were conducted to study sleep duration and dyslipidemia simultaneously or to examine their associations, especially in the Chinese population. In the present study, we investigated the association between sleep duration and serum lipid markers in a nationally representative sample, and found that shorter (⩽6 h) or longer (⩾10 h) sleep durations were associated with higher risks of abnormal lipid profiles in women. The associations were independent of age, education, occupation, current smoking, current drinking, physical activity, BMI, diabetes, and hypertension. These associations were not statistically significant in men.

Several studies explored the relationship between sleep duration and serum lipid. A recent 10-year prospective study reported that longer sleep duration was associated with a poorer lipid profile in Chicago [14]. Kaneita et al. reported a U-shaped relationship between sleep duration and a high level of TG, and between sleep duration and a low level of HDL-C in women from a National Health and Nutrition Survey in Japan [7]. Consistent with this finding, Williams et al. also found that both shorter and longer sleep duration were associated with low HDL-C in women with diabetes [15]. However, Wu et al. argued that shorter sleep duration was not associated with high TG or low HDL-C in men or in women [6]. On the other hand, Gangwisch et al. reported each additional hour of sleep duration was associated with lower risks of high cholesterol

Table 6
Association between sleep duration and serum lipid in women from the China Health and Nutrition Survey 2009 [odds ratio (95% confidence interval)].

Serum lipid	Sleep duration	Model 1[a]	Model 2[b]	Model 3[c]
High TC	⩽6	1.55 (1.24–1.92)	1.65 (1.32–2.06)	1.65 (1.32–2.06)
	7	1.18 (0.99–1.41)	1.19 (0.99–1.42)	1.19 (1.00–1.43)
	8	1	1	1
	9	1.07 (0.86–1.34)	1.11 (0.89–1.39)	1.11 (0.89–1.39)
	⩾10	1.21 (0.97–1.52)	1.27 (1.02–1.6)	1.27 (1.02–1.60)
High TG	⩽6	1.74 (1.33–2.26)	1.72 (1.30–2.26)	1.67 (1.27–2.22)
	7	1.22 (0.97–1.55)	1.21 (0.95–1.54)	1.21 (0.95–1.54)
	8	1	1	1
	9	1.28 (0.97–1.69)	1.36 (1.02–1.81)	1.34 (1.00–1.79)
	⩾10	1.82 (1.41–2.36)	1.94 (1.48–2.54)	1.96 (1.49–2.57)
High LDL-C	⩽6	1.68 (1.26–1.54)	1.72 (1.29–1.55)	1.71 (1.28–2.29)
	7	1.37 (1.06–1.47)	1.37 (1.06–1.47)	1.36 (1.05–1.76)
	8	1	1	1
	9	1.01 (0.72–1.66)	1.04 (0.74–1.67)	1.04 (0.74–1.46)
	⩾10	1.05 (0.75–1.65)	1.10 (0.79–1.65)	1.09 (0.78–1.53)
Low HDL-C	⩽6	1.22 (0.87–1.67)	1.23 (0.87–1.70)	1.22 (0.86–1.73)
	7	0.85 (0.63–1.57)	0.88 (0.65–1.59)	0.88 (0.65–1.20)
	8	1	1	1
	9	1.00 (0.71–1.69)	1.02 (0.72–1.7)	1.01 (0.71–1.44)
	⩾10	1.10 (0.78–1.68)	1.08 (0.76–1.69)	1.09 (0.77–1.55)
High ApoB	⩽6	1.08 (1.05–1.11)	1.08 (1.05–1.11)	1.80 (1.34–2.42)
	7	1.01 (0.99–1.04)	1.01 (0.99–1.03)	1.15 (0.88–1.52)
	8	1	1	1
	9	0.99 (0.97–1.02)	1.00 (0.97–1.03)	0.95 (0.66–1.35)
	⩾10	1.00 (0.97–1.03)	1.00 (0.97–1.03)	1.00 (0.70–1.43)
Low ApoA1	⩽6	2.25 (1.24–4.08)	2.04 (1.12–3.73)	2.02 (1.10–3.70)
	7	1.39 (0.79–2.42)	1.31 (0.75–2.29)	1.30 (0.74–2.28)
	8	1	1	1
	9	1.65 (0.88–3.10)	1.77 (0.94–3.33)	1.78 (0.94–3.35)
	⩾10	1.59 (0.83–3.03)	1.75 (0.91–3.38)	1.76 (0.91–3.38)
High Lp(a)	⩽6	1.01 (0.76–1.35)	1.01 (0.76–1.35)	1.00 (0.75–1.34)
	7	0.87 (0.68–1.10)	0.88 (0.69–1.12)	0.88 (0.69–1.12)
	8	1	1	1
	9	0.98 (0.74–1.30)	0.95 (0.72–1.27)	0.95 (0.72–1.27)
	⩾10	1.24 (0.94–1.62)	1.18 (0.90–1.55)	1.18 (0.89–1.55)
Dyslipidemia	⩽6	1.29 (1.04–1.60)	1.34 (1.07–1.68)	1.34 (1.07–1.68)
	7	1.01 (0.85–1.19)	1.02 (0.85–1.21)	1.04 (0.87–1.23)
	8	1	1	1
	9	0.96 (0.78–1.18)	1.03 (0.83–1.27)	1.05 (0.84–1.30)
	⩾10	1.14 (0.92–1.41)	1.22 (0.98–1.52)	1.23 (0.99–1.53)

TC, total cholesterol; TG, triglyceride; LDL-C, low density lipoprotein cholesterol; HDL-C, high density lipoprotein cholesterol; ApoB, apolipoprotein B; ApoA1, apolipoprotein A1; Lp(a), lipoprotein (a).

[a] Model 1: sleep duration, age.

[b] Model 2: sleep duration, age, education, occupation, current smoking, current drinking, physical activity.

[c] Model 3: sleep duration, age, education, occupation, current smoking, current drinking, physical activity, body mass index, hypertension, and diabetes.

in females but not in males [5]. Additionally, Bjorvatn et al. concluded that longer sleep duration was associated with lower risks of high TC and TG, but the relationship was not U-shaped in Norway [9]. In our study, a U-shaped relationship between sleep duration and serum lipids was observed only in women but not in men. The inconsistency of these results might have been influenced by the source population, lifestyle, and socio-economic status. The determination and categorization of sleep duration were also different in these studies. Sleep duration was classified into three to five groups. A continuous recording of sleep duration using objective measurement methods, such as polysomnography or actigraphy [1,16,17], was lacking. Thus, it might be difficult to study the shape of the association of sleep duration and serum lipids when treating sleep duration as a categorical variable.

ApoB is closely related to LDL-C and is regarded as an indicator and risk factor for cardiovascular diseases [18,19]. ApoA1 is a major protein component of HDL-C and is usually treated as a protective factor for cardiovascular diseases [20]. As for Lp(a), numerous studies have established its relationship with cardiovascular disease and stroke [21,22]. To the best of our knowledge, no study has been published to investigate the associations between sleep duration and ApoB, ApoA1 or Lp(a) until now. In the present study, we examined these associations in men and women separately, and found that longer sleep duration was associated with lower risks of high ApoB and ApoA1 in women, which was similar to the results for LDL-C and HDL-C.

Several potential mechanisms might be plausible to explain the statistical link of sleep duration and serum lipid. First, people with shorter sleep duration tended to show a preference for high-energy–density food, low vitamin C [23,24], and were prone to suffer weight gain via reduced leptin and increased ghrelin [25–28]. Fat-rich food, low vitamin C, and obesity are risk factors for dyslipidemia [29,30]. In the present study, the associations of sleep duration and lipid biomarkers were only slightly changed after adjusting for BMI. This means that BMI or obesity might contribute little to the relationship between sleep duration and abnormal lipids. Second, energy expenditure was reported to be lowered in those with short sleep duration [31]. Third, sleep duration and abnormal lipids share some common risk factors, such as smoking [32], alcohol [33], and lower socio-economic status [34]. Thus, the neuroendocrine system, unhealthy lifestyle, and disadvantaged socio-economic position might present a biological explanation for sleep duration and abnormal lipids. However, the associations changed only a little after adjusting for smoking, alcohol drinking, and socio-economic status in our study. These findings were suggestive of other causal pathways to explain the observed associations.

Although the sample size of the present study is large, there are some limitations that need to be considered when interpreting the results. First, a causal relationship between sleep duration and lipid profiles cannot be determined in our study due to its cross-sectional design. Furthermore, short sleep duration was reported to be associated with stress [35], which is also a risk factor for abnormal lipids [36,37]. In this study, there was no measurement of stress and therefore we could not adjust for its potentially confounding effect. Finally, the measurement of sleep duration was via self-reported questionnaire and the direction and degree of self-report bias for sleep duration remains to be evaluated. Future longitudinal studies would ideally include objective measures of sleep and control for more covariates such as stress.

5. Conclusions

Self-reported short and long sleep durations were significantly associated with a poorer lipid profile in women, whereas these associations were not significant in men.

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Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2014.02.006.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.sleep.2014.02.006.

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